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HUMAN PAROTID GLAND ALPHA-AMYLASE SECRETION AS A FUNCTION OF CHRONIC HYPERBARIC EXPOSURE

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Human parotid gland alpha-amylase secretion as a function of chronic hyperbaric exposure

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Gilman, S. C., G. J. Fischer, R. J. Biersner, R. D. Thornton, and D. A. Miller. 1979. Human parotid gland alpha-amylase secretion as a function of chronic hyperbaric exposure. Undersea Biomed. Res. 6(3):303–307.—Secretion of α-amylase by the human parotid gland increased significantly during eight days of hyperbaric exposure. This hyperactivity of the parotid gland presumably resulted from increased autonomic nervous system (ANS) activity attributable to (1) psychological stress in the form of anticipation; (2) dive-related factors, i.e., hyperoxia, P_{N_2}, physical stress; or (3) a combination of both. The etiology of the effect must await additional studies, but a consistent and significant elevation in α-amylase secretion was found. This previously undescribed effect of hyperbaric exposure indicates that parotid α-amylase sampling holds promise as a noninvasive means of monitoring physical and psychological stress, and as an indirect measure of ANS tone.

stress
autonomic nervous system
hyperoxia
α-amylase

Various types of physical and psychological stressors have been found to cause a marked autonomic nervous system (ANS) response, with numerous effects in various target organs attributable to activation of adrenergic receptors. Stressors evoking this increased ANS activity include problem-solving under time pressure (Frankenhaeuser and Patkai 1965), space and supersonic flight (Goodall, McCally, and Graveline 1964), hazardous military duty (Bloom, von Euler, and Frankenhaeuser 1963), *g* force in a centrifuge (Silverman and Cohen 1960), and cold-water immersion (Speirs, Herring, Cooper, Hardy, and Hind 1974).

Like other stressors, factors associated with chronic exposure to hyperbaric pressure, e.g., hyperoxia, increased P_{N_2}, might similarly increase ANS activity. Such increased ANS activity could have detrimental effects on divers under hyperbaric exposure (Bean and Johnson 1955; Pagni, Zampolini, and Frullani 1967; Geiger, Brumleve, Boelkins, and Parmar 1976). Unfortunately, it is difficult both technically and operationally to monitor target organ re-

sponse to ANS activity. An exception might be secretory activity by the parotid gland. The present study assessed the possibility that secretion by the parotid gland might prove to be a reliable indicator of the ANS stress response to hyperbaric exposure. This was done by monitoring parotid gland activity in 11 United States Navy divers before, during, and after eight days in a hyperbaric chamber.

METHODS

Since the capacity of the hyperbaric chamber was limited to 3 or 4 divers, divers were run in successive squads of 4, 4, and 3, for a total of 11 subjects in the experiment. They were all adult male United States Navy divers to whom the entire experiment and everything that would happen to them had been explained eight days before the experiment began. After that explanation, all had the option to decline to participate, but none did. The divers were exposed to hyperbaric air for eight days in a 10 × 30 ft steel hyperbaric chamber maintained at the Naval Submarine Medical Research Laboratory. The saturation pressure was 2.8 ATA (equivalent to 60 ft of seawater (fsw)), with daily 8-h excursions (starting at 10 a.m.) to 4.0 ATA (equivalent to 100 fsw). These excursions did not require decompression on return to 2.8 ATA (60 fsw). A recirculation atmosphere control system was used to regulate Po_2 and Pco_2 at 20.9% and <1%, respectively. Temperature and humidity were regulated for diver comfort. The divers' daily schedule included numerous physiological and behavioral tests commencing at 10 a.m. and ending at 8 p.m. daily. Decompression to the surface required 20 h and began at 2 p.m. on the seventh day.

The subjects were placed in the chamber for one hour daily for 8 days prior to the dive and for 2 (the second and third) days after the dive. Parotid samples were obtained from the subjects daily at 6 a.m. on these days, as well as on the 8 days during the dive (including decompression). Stimulated parotid fluid was collected for 3 min using a new zero priming volume collector that does not require the use of a positioner.[1] Sour grape candy, a standard stimulus, was used to elicit the secretion. After collection, the fluid was frozen for subsequent α-amylase measurement. Within one week after collection, α-amylase levels were determined using the amylochrome method (Roche).

RESULTS

The mean rate of α-amylase secretion per day within pre-dive, dive, and post-dive periods is shown in Fig. 1. For purposes of visual comparison, base-line data for a one-week period also are shown for 6 Navy divers from another experiment. Their α-amylase secretion was measured daily and similarly, except that the divers were not scheduled for a dive. Means in the present study were assessed using each diver's (total of 11 subjects) days-within-time-periods (pre-dive, dive, and post-dive) repeated measurements' design analysis of variance. Results revealed significant differences between time periods [$F(2,20) = 8.54; P < 0.01$] and between days within the pre-dive and dive time periods [$F(7,150) = 3.07$ and 3.18, respectively; $P < 0.01$]. The nature of the difference between time periods can be seen in Fig. 1. Specifically, α-amylase secretion during the dive was significantly higher than during the pre-dive or post-dive time periods, which did not differ from each other. Within the pre-dive period α-amylase increased significantly by Day 3 [for Day 3 vs. Days 1 and 2: $F(1,150) = 4.65$;

[1]A description of the new parotid fluid collector may be obtained from the third author.

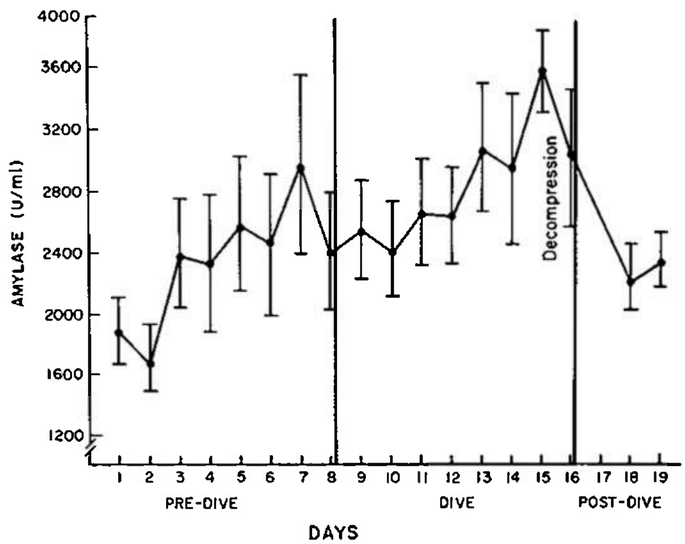

Fig. 1. Parotid gland α-amylase secretion before, during, and after eight days of hyperbaric exposure; •--- • represents control values.

$P < 0.05$], continued to increase gradually through Day 7, and then declined. For example, α-amylase secretion on Day 7 was not significantly greater than on the previous day [F (1, 150) = 3.26; $P > 0.05$], but was significantly greater than on the following day, Day 8 [F (1, 150) = 4.21; $P < 0.05$]. An essentially linear increase in α-amylase secretion as the dive approached could be interpreted as anticipatory stress. However, the decline two hours before entering the chamber (Day 8) would not be accounted for on this basis. During the dive, the significant difference between mean α-amylase secretion over days was simply that α-amylase increased linearly (see Fig. 1). The increase was statistically significant from Day 14 to Day 15, the day before decompression [for Day 14 vs. Day 15, F (1,150) = 4.78; $P < 0.05$]. This result is consistent with the hypothesis of anticipatory stress. So is the decline in α-amylase on Day 16. This is because stress, and thus α-amylase secretion, would be expected to decline once the divers were nearly through with decompression, the most dangerous part of the dive. Finally, α-amylase secretion declined significantly from the last dive day to the second and third post-dive days [for Day 16 vs. Day 19 (and thus Day 18), F (1,150) = 5.10; $P < 0.05$].

DISCUSSION

The linear increase in secretion of α-amylase found during eight days of hyperbaric exposure is notable in view of previous research suggesting that this α-amylase secretion is a direct result of adrenergic nerve activation by catecholamines (CA) (Strömblad and Nickerson 1961; Heidel 1967; Batzri, Selinger, Schramm, and Robinovitch 1973). For example, stimulation of the adrenergic system with catecholaminergic agents causes marked release of α-amylase (Batzri et al. 1973; Butcher, Goldman, and Nemerovaski 1975). This increased secretion of α-amylase is blocked by the adrenergic antagonist, propranol (Schramm and Naim 1970). *In vitro* studies of parotid gland slices show that activation of adrenergic receptors, or addition of CA to the slice media, causes almost total secretion of the accumulated α-amylase (Batzri and

Selinger 1973). Also, at least one other stressful situation known to increase ANS activity and CA secretion, cold water immersion, has been found to produce an increase in α-amylase secretion (Speirs et al. 1974).

The elevation of α-amylase secretion found before the dive could have been due in part, at least, to anticipatory stress. During the dive, continued elevation of α-amylase could have been due to anticipatory stress or other psychological factors, e.g., confinement, sensory deprivation, fatigue. However anticipatory stress, but not other psychological factors, would account for the significant elevation on the day just before decompression and the decline the next day when decompression was nearly complete. Anticipatory stress may even account better for the findings than physiological factors. For example, the absence of toxic effects, such as vital capacity changes, throughout the dive indicates that the significant rise just before decompression was not related to oxygen toxicity or other physiological factors. Also, parotid secretion of α-amylase was still high five hours before the divers left the chamber, when they were only a few feet from the surface. Finally, similar anticipatory effects using other measures of ANS activity, e.g., catecholamine excretion, have been noted in previous research (Demos, Hale, and Williams 1969; Mason 1975; Frankenhaeuser 1971, 1975). Thus, present findings support that anticipatory stress plays a role during hyperbaric exposure and that this stress may be detected via a physiological response, i.e., increased α-amylase secretion. Further, parotid α-amylase secretion seems a valid indicator of ANS activity, though the relation of psychological factors to α-amylase secretion during stress situations, such as diving, should be documented further.

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Gilman, S. C., G. J. Fischer, R. J. Biersner, R. D. Thornton, and D. A. Miller. 1979. La sécrétion d'alpha-amylase de la glande parotide chez les humains comme fonction de l'éxposition chronique hyperbare. Undersea Biomed. Res. 6(3):303–307.—La sécrétion d'α-amylase de la glande parotide chez les humains ont augmenté significantivement pendant huit jours de l'éxposition hyperbare. Cette hyperactivité de la glande parotide a présumablement résulté de l'activité augmentée du système nerveux autonome (SNA) attribuée à (1) la tension psychologique de l'anticipation; (2) les facteurs qui se rapportent à la plongée, i.e., l'hyperoxie, PN_2, la tension physique; ou (3) une combinaision de tous les deux. L'etiologie de l'effet doit attendre les résultats des études supplémentaires, mais une élévation significante et consistante de la sécrétion d'α-amylase a été découverte. Cet effet de l'éxposition hyperbare, qui auparavant n'a pas été décrite, indique que le prélèvement du parotid α-amylase donne des espérances d'être un moyen non-invasif de contrôller la tension physique et psychologique, et d'être une mesure indirecte du ton de SNA.

tension
systeme nerveux antonome
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